

1:20-CV-98

Plaintiff's motion to exclude quantification of relative asbestos fiber potency, (ECF No. 253), will be granted. Plaintiff's motion to exclude "No Observed Adverse Effect Level" testimony, (ECF No. 255), will be granted.

## **I. BACKGROUND<sup>1</sup>**

Mr. Walls served in the Navy from 1955 to 1959 and then worked as a tractor-trailer<sup>2</sup> fleet mechanic for approximately 40 years from 1960 to 2002 in North Carolina and Virginia. (ECF No. 488 at 2.) As a fleet mechanic, Walls performed maintenance on tractor-trailer brakes, clutches, and engine gaskets manufactured by Defendants. (*Id.* at 2–4.) These products all contained asbestos until the 1970s, and some contained asbestos into the 1990s. (*Id.* at 4–5.) Walls was diagnosed with mesothelioma—a form of lung cancer—on September 8, 2019, and died from the disease on October 15, 2020. (*Id.* at 5.)

Plaintiff and her husband filed this suit on January 30, 2020, against nineteen Defendants. (ECF No. 1.) Several Defendants have since been dismissed from this action. On February 25, 2022, this Court ruled on Plaintiff's and Defendants' motions for summary judgment. (ECF Nos. 487; 488.) Defendant Ford has moved the Court to reconsider its Order granting in part and denying in part Ford's motion for summary judgment, (ECF No. 512), and that motion is pending before this Court. This matter is set for jury trial on October 3, 2022. (ECF No. 486.)

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<sup>1</sup> A full discussion of the facts and evidence in this case is available in this Court's dispensation of the parties' motions for summary judgment. (ECF Nos. 487 at 4–7; 488 at 2–5.)

<sup>2</sup> Tractor trailer trucks are commonly known as semi-trailers or eighteen wheelers. (ECF No. 488 at 2 n.1.) The truck is the "tractor" and the cargo box or platform it tows is the "trailer." (*Id.*)

Plaintiff and the remaining Defendants filed approximately twenty-five *Daubert* motions to exclude expert testimony.<sup>3</sup> On March 16, 2022, this Court held a teleconference with counsel for the parties to discuss certain matters to include the need to eliminate duplication among the motions, clarification of the specific issues to be addressed, and the process by which a hearing related to this volume of motions could proceed most efficiently and effectively. (ECF No. 494.) Defendants subsequently moved to join Defendant Ford’s *Daubert* motions and briefing, thereby “obviate[ing] the need for the Court to issue separate rulings on” Defendants’ remaining motions. (ECF No. 499 at 2; *see also* ECF Nos. 500–05.) This Court granted Defendants’ motions to join on June 7, 2022. (ECF No. 512.) On June 15 and 16, 2022, this Court heard oral argument from the parties on the remaining *Daubert* motions. (ECF No. 522.) The motions are now fully briefed and argued and are ripe for decision.

## **II. STANDARD OF REVIEW**

The admissibility of expert opinion is governed by Rule 702 of the Federal Rules of Evidence and the Supreme Court’s landmark ruling in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). Rule 702 provides that a witness “who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:”

- (a) the expert’s scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
- (b) the testimony is based on sufficient facts or data;
- (c) the testimony is the product of reliable principles and methods; and

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<sup>3</sup> *See* ECF Nos. 237; 239; 243; 251; 253; 255; 256; 258; 262; 264; 269; 273; 277; 279; 281; 297; 301; 303; 305; 307; 308; 309; 329; 333.

- (d) the expert has reliably applied the principles and methods to the facts of the case.

Fed. R. Evid. 702. Thus, expert testimony is admissible only if: (1) the expert is qualified, (2) the testimony is relevant, and (3) the testimony is based on reliable scientific methodology.<sup>4</sup> See *Daubert*, 509 U.S. at 594–95. The Court must find these elements “at the outset, . . . by a preponderance of proof.” *Id.* at 592, 592 n.10.

An expert is *qualified* if he or she has “specialized knowledge that will assist the trier of fact in understanding the evidence or determining a fact in issue.” *United States v. Young*, 916 F.3d 368, 379 (4th Cir. 2019). A witness’ qualifications are “liberally judged by Rule 702,” and “a person may qualify to render expert testimony in any one of the five ways listed” by the Rule: “knowledge, skill, experience, training, or education.” *Kopf v. Skyrms*, 993 F.2d 374, 377 (4th Cir. 1993); see *Cooper v. Lab’y Corp. of Am. Holdings*, 150 F.3d 376, 380 (4th Cir. 1998).

An expert who is qualified must provide testimony that is relevant. An expert’s opinion is *relevant* if it “fit[s]” the facts of the case, meaning it has “a valid scientific connection to the pertinent inquiry.” *Daubert*, 509 U.S. at 591–92. “This ensures that the expert ‘helps the trier of fact to understand the evidence or to determine a fact in issue.’” *Sardis v. Overhead Door Corp.*, 10 F.4th 268, 281 (4th Cir. 2021) (quoting *Nease v. Ford Motor*

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<sup>4</sup> Although *Daubert* interpreted an earlier version of Rule 702, “the standard of review that was established for *Daubert* challenges is still appropriate” to assess the admissibility of expert testimony. *United States v. Parra*, 402 F.3d 752, 758 (7th Cir. 2005); see *In re Viagra (Sildenafil Citrate) & Cialis (Tadalafil) Prod. Liab. Litig.*, 424 F. Supp. 3d 781, 789 (N.D. Cal. 2020) (“[N]o obvious conflict arises between [Rule 702] as amended and *Daubert*, at least as relevant to the issues in this case.”); see also *Sardis v. Overhead Door Corp.*, 10 F.4th 268, 282 (4th Cir. 2021) (“Rule 702 was amended specifically to affirm the trial courts role as gatekeeper.” (internal quotations omitted)).

*Co.*, 848 F.3d 219, 229 (4th Cir. 2017)). “Simply put, if an opinion is not relevant to a fact at issue, *Daubert* requires that it be excluded.” *Id.* at 281.

Finally, relevant testimony must also be reliable. An expert’s opinion is *reliable* if it is “based on scientific, technical, or other specialized knowledge and not on belief or speculation.” *Id.* (emphasis omitted) (quoting *Oglesby v. Gen. Motors Corp.*, 190 F.3d 244, 250 (4th Cir. 1999)). While the subject of scientific testimony must not “be ‘known’ to a certainty,” it must be “derived by the scientific method” and “supported by appropriate validation—*i.e.*, ‘good grounds,’ based on what is known.” *Daubert*, 509 U.S. at 590. Reliability is a “flexible” inquiry that must focus “solely on principles and methodology, not on the conclusions that they generate.” *Id.* at 594–95. In *Daubert*, the Court outlined a non-exhaustive list of factors to guide lower courts in assessing reliability, including: (1) whether the theory can be (and has been) tested; (2) whether it has been subjected to peer review and publication; (3) its potential rate of error; (4) whether standards exist to control the technique’s operation; and (5) the degree of acceptance of the methodology within the relevant scientific community. *Id.* at 593–94. These factors “may or may not be pertinent in assessing reliability, depending on the nature of the issue, the expert’s particular expertise, and the subject of his testimony,” and courts have “broad latitude” in choosing which factors are “reasonable measures of reliability in a particular case.” *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 150, 153 (1999).

“Expert evidence can be both powerful and quite misleading because of the difficulty in evaluating it. Because of this risk, the judge . . . exercises more control over experts than over lay witnesses.” *Daubert*, 509 U.S. at 595. Rule 702 “imposes a special gatekeeping obligation on the trial judge to ensure that an expert’s testimony both rests on

a *reliable* foundation and is *relevant* to the task at hand.” *Sardis*, 10 F.4th at 281 (internal quotations omitted). A court cannot “abandon the gatekeeping function” by deferring its responsibility to the jury. *Id.* at 282 (quoting *Kumho*, 526 U.S. at 159 (Scalia, J., concurring)). Ultimately, a district court’s Rule 702 analysis “necessarily amount[s] to an exercise of broad discretion guided by the overarching criteria of relevance and reliability.” *Belville v. Ford Motor Co.*, 919 F.3d 224, 233 (4th Cir. 2019) (quoting *Oglesby*, 190 F.3d at 250).

Although Rule 702 “is not intended to serve as a replacement for the adversary system,” *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prod. Liab. Litig. (No II)* MDL 2502, 892 F.3d 624, 631 (4th Cir. 2018), this Court takes seriously its gatekeeping role to protect lay jurors from “powerful and quite misleading” expert testimony, *Daubert*, 509 U.S. at 595. The Court will address each motion to exclude expert testimony in turn.

### **III. DEFENDANTS’ MOTIONS (ECF Nos. 307; 308; 309)**

Defendants move to exclude the testimony of Dr. Murray Finkelstein, Dr. Edwin Holstein, and Dr. John Maddox. (ECF Nos. 307; 308; 309.) Defendants do not challenge Finkelstein’s, Holstein’s, or Maddox’s qualifications. (June 16 Tr. at 12:7-10.) Rather, Defendants argue that these experts’ general causation opinions—that chrysotile asbestos from vehicle friction products can cause mesothelioma—and specific causation opinions—that Defendants’ products caused Walls’ mesothelioma—are unreliable and do not fit the facts of this case. (ECF No. 477 at 1.)

The Court finds that Plaintiff’s experts are qualified to offer the challenged opinions in this case. Finkelstein is a physician and epidemiologist with over forty years of experience. (ECF No. 479-49 at 1–3.) He holds a master’s degree and Ph.D. in Physics from Case-Western Reserve University and a *Medicina Doctorem et Chirurgia Magistrum*

(M.D.C.M.) degree from McGill University. (*Id.* at 1.) He currently serves as an Associate Professor of Occupational Health and Environmental Medicine at McMaster University and an Assistant Professor of Family and Community Medicine and Public Health at the University of Toronto. (*Id.* at 1–2.) He has published over one hundred scholarly articles in peer-reviewed journals. (*Id.* at 11–22.)

Holstein is a Clinical Assistant Professor in the Division of Environmental Sciences at Mount Sinai School of Medicine and a licensed physician with nearly fifty years of experience. (ECF No. 479-47 at 1–2.) He holds a Master of Science from Massachusetts Institute of Technology and an M.D. from Mount Sinai School of Medicine. (*Id.* at 2.) He is board certified in Internal Medicine and Preventive Medicine, with a specialty in Occupational Medicine. (*Id.*) He has dedicated much of his career to the study and prevention of asbestos-related diseases. (*See id.* at 2–6.) He has published several scholarly articles and presentations on asbestos. (*Id.* at 4–6.)

Maddox is a pathologist with board certification in Anatomic and Clinical Pathology. (ECF No. 479-50 at 1.) He holds an M.D. from the University of Virginia School of Medicine. (*Id.*) In 2019, he retired after thirty years as a clinical pathologist at Riverside Regional Medical Center in Newport News, Virginia, where he saw hundreds of cases of mesothelioma. (*Id.*) He has published several scholarly articles. (*Id.* at 2.)

Plaintiff's experts offer four conclusions concerning general and specific causation relevant to Defendants' motions: (1) mesothelioma is a dose-response disease caused by the cumulative dose of asbestos, (2) chrysotile asbestos in vehicle friction products can cause mesothelioma, (3) exposure to each Defendant's products contributed to Walls' mesothelioma, and (4) each Defendant's products constituted a "substantial cause" of

Walls' mesothelioma under North Carolina law. (Finkelstein rep. at 11; Holstein rep. III at 7; Maddox rep. at 75.) The Court will address each opinion in turn.

#### **A. General Causation**

Mesothelioma is a cancer in the mesothelial cells of the pleura—the lining of the lungs. (Maddox rep. at 11.) It occurs when a mesothelial cell's DNA mutates and becomes carcinogenic. (*Id.* at 15.) Mesothelioma is “an incurable and uniformly fatal cancer.” (*Id.*) According to Plaintiff's experts, asbestos is the “only relevant known cause of mesothelioma.” (Finkelstein rep. at 26; Maddox rep. at 15; Holstein rep. I at 28.) Where a person without any known asbestos exposure contracts mesothelioma, her cancer is considered “idiopathic,” meaning it has an unknown cause. (Holstein rep. I at 28.) Holstein estimates that approximately ninety-five percent of mesotheliomas in the United States are caused by known exposure to asbestos. (*Id.* at 29.)

The body has tools to prevent mesothelioma, and not everyone who is exposed to asbestos develops cancer. (Maddox rep. at 13–14.) Some asbestos fibers are filtered by hairs in the nose, captured in mucus, coughed up, or destroyed by macrophages or “scavenger cells” that destroy foreign objects in the lungs. (*Id.* at 13.) Where asbestos fibers do reach the pleura, the body's immune system works to kill mesothelioma cells that have mutated due to asbestos exposure. (*Id.* at 45–46.) “There is a substantial individual susceptibility component to asbestos-induced malignancies.” (*Id.* at 36.) “Some individuals develop mesothelioma following exposure to small amounts of asbestos, whereas others exposed to heavy amounts do not.” (*Id.*) As a consequence of the body's ability to prevent mesothelioma, “as many as 90% of people with huge asbestos exposures . . . never develop mesothelioma.” (*Id.* at 37.)



Plaintiff's experts testify that mesothelioma is a dose-response disease, meaning that "the greater the dose of asbestos, the greater the risk for developing mesothelioma." (Maddox rep. at 37; Holstein rep. I at 26; Finkelstein rep. at 25.) They characterize the dose-response relationship as linear, meaning that "the number of people who develop mesothelioma increases in proportion to the extent of exposure of populations to asbestos." (Maddox rep. at 3.) "This dose response curve also applies to individuals," meaning that the greater the dose, the greater the likelihood that an individual will develop mesothelioma, even at low levels of exposure. (*Id.*)

While Holstein reports that it is "theoretically possible" that a single fiber could cause mesothelioma, (Holstein rep. I at 27), Plaintiff's experts agree that "it is the total dose of asbestos that the patient breathes that is the cause of the disease," (*id.*; Maddox rep. at 2 ("[Mesothelioma] is not caused by a single fiber or even a single exposure, but rather by the inhalation of millions of microscopic asbestos fibers over time.")). "[T]here are fibers from each exposure that make their way to the pleura," (Holstein rep. I at 29), and these multiple exposures work together to cause mesothelioma by (1) creating multiple genetic mesothelial cell defects throughout the lungs, (2) suppressing the body's immune system response to mutated cells, and (3) reducing the latency period, (Maddox rep. at 3). "There is an inverse relationship between the amount of asbestos a person inhales and the time period in which one of these cancers will develop," meaning higher cumulative asbestos-fiber dose "lead[s] to an earlier development of mesothelioma." (Holstein rep. I at 26–27.) In other words, "[t]he cumulative exposure to asbestos that a mesothelioma victim has received in his/her lifetime has caused impact to the lungs and pleura, has overwhelmed the body's defense mechanisms, and has caused that mesothelioma." (*Id.* at 41.)

Plaintiff's experts testify that "[i]t is widely reported in scientific, medical, and government reports that very low exposures to asbestos, e.g. days and weeks, have been attributed as the cause of mesothelioma, and scientists have not been able to determine a threshold of minimal exposure below which numerous mesotheliomas will not occur." (Maddox rep. at 3.) They report that "[t]he majority of the scientific community holds the opinion that there is no known level of asbestos exposure above ambient air levels which has not been shown to contribute to the development of mesothelioma in a sufficiently large, exposed population." (Holstein rep. I at 31; Finkelstein rep. at 25 ("There is no known safe level of asbestos exposure established for the prevention of mesothelioma.").)

Mesothelioma rates also vary by type of asbestos exposure. Asbestos is a naturally occurring mineral that is mined and included in various friction products. (June 15 Tr. at 105:4-6.) There are six types of asbestos, the first being chrysotile, and the remaining five being amphibole asbestos. (*Id.* at 104:23–105:2.) Chrysotile shards are smaller than amphibole shards, have a different shape chemical structure, and (unlike amphibole shards) do not contain iron. (*Id.* at 22:12-18.) Defendants' products all used chrysotile asbestos, although each likely contained small amounts of tremolite, an amphibole that grows in the same mineral deposits as chrysotile and cannot be effectively filtered. (*Id.* at 105:3-10.)

The parties agree that chrysotile asbestos is less potent than amphibole asbestos. (*Id.* at 130:8-11.) Nevertheless, Maddox testifies that chrysotile asbestos is a genotoxic carcinogen, meaning it can cause cancerous mutations in a cell's genes. (Maddox rep. at 45.) Chrysotile asbestos "is involved in both direct tumor generation (initiation) and indirect tumor growth (promotion)." (*Id.*) Chrysotile can directly initiate cancer by mutating genes that "lead to a cancerous tumor in the first place." (*Id.*) It can also indirectly

promote cancer by inflaming the lungs, which “stimulate[s] cell proliferation and cause[s] inflammation and suppression of the body’s immune response and ability to kill mutant cells.” (*Id.* at 46.) Thus, Maddox testifies, each exposure to millions of chrysotile fibers contributes to the cause of mesothelioma by creating mutations, stimulating proliferation of mutant cells, or suppressing the body’s ability to kill them. (*Id.*)

Defendants contend that there is also a difference between mined chrysotile asbestos and “manufactured chrysotile,” or chrysotile that has gone through the vehicle friction product manufacturing process. (ECF No. 477 at 18–20.) When chrysotile is heated to extreme temperatures, it loses its water molecules, loses its chemical structure, and becomes non-toxic. (*Id.* at 18.) Defendants cite a mineralogy study that found chrysotile also begins to lose its potency when heated to temperatures reached during manufacturing, an animal study that showed inflammation in rats exposed to “manufactured chrysotile” went away one year after exposure, and epidemiology studies and meta-analyses that found no increased risk of mesothelioma among vehicle mechanics. (*Id.* at 20.) From these studies, Defendants argue that “manufactured chrysotile” does not cause mesothelioma. (*Id.* at 18–20.)

Maddox does not offer a categorical opinion about the potency of chrysotile fibers from vehicle friction products, but both Finkelstein and Holstein testify that chrysotile from friction products can cause mesothelioma. Finkelstein reports that “[t]here is overwhelming, generally accepted evidence that inhalation of asbestos fibers of any type, from any source or product, may cause mesothelioma.” (Finkelstein rep. at 11.) He supports this opinion with citations to fiber burden studies of brake mechanics that found that “asbestos fibers are deposited and retained in the lungs of brake mechanics,” (*id.* at 20),

as well some epidemiologic studies of mechanics that, according to his analysis, show an increased risk of mesothelioma among vehicle mechanics, (*id.* at 23–25). Holstein similarly testifies that “[t]he causal relationship between exposure to chrysotile asbestos and mesothelioma or lung cancer has been observed across several different cohorts of workers exposed to chrysotile,” including vehicle mechanics. (Holstein rep. I at 36–37; Holstein rep. III at 3–4 (citing a study that found that “workers in industries and occupations related to motor vehicle manufacturing and repair, such as motor vehicle mechanics . . . had elevated rates of mesothelioma.”).) He cites case studies of patients with mesothelioma where only chrysotile asbestos was found in the patients’ lungs. (Holstein rep. I at 37.) And he cites animal studies where chrysotile was injected directly into the animal’s pleura and subsequently caused malignant changes in the mesothelial cells. (*Id.* at 38.) Both experts explicitly consider opposing epidemiological evidence in forming their opinions but concluded that those “[p]ublished epidemiologic studies are generally not helpful” because “individual exposure information is not available and the number of workers at risk is surely overestimated.” (Finkelstein rep. at 22–23; Holstein rep. I at 44–45.) They report that these studies lump auto workers together regardless of type of vehicle work or cumulative asbestos exposure. (Holstein rep. I at 45; Finkelstein rep. at 22–23.)

Based on the preponderance of the evidence, the Court finds that these general causation opinions are relevant and based on reliable scientific methodologies. First, it appears that all experts from both sides agree that mesothelioma is a dose-response disease caused by the total exposure to asbestos over a person’s lifetime. (*See, e.g.*, Crapo rep. at 6–7 (“Scientific research has established that the effect of exposure to asbestos fibers in causing disease is related to the dose of asbestos delivered . . . and the tissue reactions that

occur when large numbers of fibers accumulate over time due to consistently high levels of exposure.”).) Defendants’ experts contend that the dose-response curve is non-linear at low levels of asbestos exposure, and that “simple linear extrapolation at low doses may result in an overestimation of cancer risk.” (Mowat rep. at 35.) However, the Court finds that Holstein’s and Maddox’s theory that the dose-response curve remains linear even at low levels of exposure is “supported by appropriate validation—*i.e.*, ‘good grounds,’ based on what is known.” See *Daubert*, 509 U.S. at 590. This theory has been repeatedly tested and, according to Holstein’s and Maddox’s analysis, no examination of low levels of asbestos exposure has proven a safe dose. (See Holstein rep. I at 31–34.) Their extrapolation has been repeatedly reviewed and published and appears to be accepted within the relevant scientific community, including by the Environmental Protection Agency, Occupational Safety and Health Administration, National Institute for Occupational Safety and Health, and World Health Organization. (*Id.*)

Second, Finkelstein’s and Holstein’s opinion that chrysotile in vehicle friction products can cause mesothelioma is reliably based on their review of epidemiological research and case studies showing that vehicle mechanics are at a higher risk of contracting mesothelioma. Moreover, while Defendants have offered scientific evidence that chrysotile asbestos in vehicle friction products may be less potent than other forms of asbestos, this evidence does not show that chrysotile asbestos loses *all* potency during manufacturing. (June 15 Tr. at 73:23–74:10 (conceding that whether chrysotile fibers lose all potency is “the missing piece”).) And Defendants’ animal studies showing a lack of inflammation after one year are not definitive as those studies did not examine whether rats later developed cancer. Holstein and Finkelstein have used reliable scientific methods to opine that chrysotile

asbestos causes cancer at low levels and that vehicle mechanics are at increased risk of developing mesothelioma. Thus, even if “manufactured chrysotile” is less potent, Holstein and Finkelstein may still reliably opine that chrysotile from vehicle friction products can cause cancer generally.

## **B. Specific Causation**

According to Holstein, mesothelioma is “a ‘signal tumor’ for asbestos exposure,” meaning that, “[b]ecause asbestos dust is so strongly associated with mesothelioma, proof of significant exposure to asbestos dust is proof of specific causation.” (Holstein rep. I at 26, 28.) “As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and in so doing shortens the necessary period for asbestos disease to develop, each significant exposure to asbestos contributes to the development of the malignant mesothelioma or lung cancer that actually occurred, when it occurred, in a given patient.” (*Id.* at 27.) “[T]he best scientific evidence is that all significant exposures contribute to the causation of a subsequent mesothelioma or lung cancer.” (*Id.*) Holstein defines an exposure as significant if it was “at a level where disease has occurred in other settings.” (*Id.* at 30–31.) According to him, “even brief or low-level exposures” may be “significant.” (*Id.* at 31.)

Applying this analysis, Holstein considered in detail Plaintiff’s exposure to each Defendant’s products and concluded that each contributed to his mesothelioma. (Holstein rep. III at 5–18.) He based these conclusions on a thorough review of Walls’ deposition testimony, in which he describes in detail his work with each Defendant’s products, and a report prepared by another of Plaintiff’s experts, Christopher DePasquale, an industrial hygienist, among other documents. (*Id.* at 1, 5–18.) DePasquale reports that removing,

cleaning, and replacing brakes as described by Walls can cause “significant airborne asbestos exposures [to] occur to the mechanic.” (*See* ECF No. 415-10 at 22 (DePasquale rep.).) Exposure is measured in fibers per cubic centimeter of air (“f/cc”). (*Id.*) According to DePasquale, a mechanic’s exposure to asbestos while cleaning brake drums with compressed air can range from 6.6 to 29.8 f/cc, and peak concentrations of 87 f/cc can occur. (*Id.*) Filing down new brakes can cause exposure ranging from 0.1 to 0.9 f/cc. (*Id.* at 24.) Cleaning and replacing clutches can cause personal exposure of 2.25 f/cc, and scraping asbestos engine gaskets can create concentrations up to 2.6 f/cc. (*Id.* at 25–26.) Sweeping asbestos dust at the end of a work shift “has been shown to cause exposures” of up to 1.7 f/cc. (*Id.* at 23.) One study cited by DePasquale found that merely opening boxes that contain new brakes can expose airborne fiber concentration of up to 1.9 f/cc. (*Id.* at 24.)

Relying on these estimates, Holstein concludes that Walls engaged in activities with each Defendant’s products on a “frequent, regular and recurring basis” that created high concentrations of airborne asbestos which he inhaled, causing significant exposures. (Holstein rep. III at 5–18.) Given his opinion that every significant exposure contributes to the development of mesothelioma either by mutating new mesothelial cells, stimulating proliferation of those cells, or suppressing the body’s ability to kill mutated cells, Holstein opines that exposure to each Defendant’s products contributed to Walls’ mesothelioma. (*Id.*)

Finally, Holstein testifies that exposure to each Defendant’s products constituted a “substantial factor” in the causation of Walls’ mesothelioma. (*Id.* at 7–17.) “Substantial factor” is a legal term, not a scientific term. To show causation in an asbestos suit in North

Carolina, a plaintiff must prove (1) “that he was actually exposed to the alleged offending products,” *Smith v. Schlage Lock Co., LLC*, 986 F.3d 482, 487 (4th Cir. 2021) (quoting *Wilder v. Amatex Corp.*, 336 S.E.2d 66, 68 (N.C. 1985)), and (2) “that exposure to a defendant’s product was a substantial factor causing the plaintiff’s injury,” *Finch v. Covil Corp.*, 972 F.3d 507, 512 (4th Cir. 2020). Whether exposure to a particular defendant’s product constituted a “substantial factor” is a question for the jury to answer after considering the frequency, regularity, amount, and proximity of exposure, *id.* at 512–13, as well as alternative potential causes, *Connor v. Covil Corp.*, 996 F.3d 143, 155 (4th Cir. 2021). An exposure to asbestos may be medically “significant” in that equivalent exposures have been shown to cause mesothelioma in other cases but still not be considered a “substantial factor” if, based on the specific facts of the case, the exposure is “dwarfed” by a “far more frequent, regular, and close-proximity exposure.” *Id.* Here, Holstein recites the relevant legal factors and opines that Walls’ “frequent, regular, and proximate exposures . . . each taken alone, constituted a substantial factor in the causation of Mr. Walls’ malignant mesothelioma.” (Holstein rep. I at 9–10; Holstein rep. III at 5–18.)

Maddox offers similar testimony. He considers exposure to a particular defendant’s product to contribute to cause mesothelioma if (1) the cumulative exposure is sufficient to cause mesothelioma, and (2) the exposure to the particular defendant’s product was also “sufficient to cause mesothelioma.” (Maddox rep. at 30.) He does not consider every exposure that contributes to a person’s cumulative dose to constitute a “cause.” (*Id.* at 74.) Rather, an exposure is “sufficient to cause mesothelioma” if it was “sufficient in and of itself to cause a risk of malignant mesothelioma,” meaning it included “significant, repetitive occupational *exposures* to asbestos that are at least several orders of magnitude greater than



background ambient air exposure levels, and which occur over a significant duration of time in close proximity to the worker.” (*Id.* at 30, 73.) This is a “qualitative assessment” that involves reviewing:

the nature of exposure, the level of exposure and the duration of exposure, whether a product releases significant respirable asbestos fibers during its intended use, the approximate percentage of asbestos in the product, . . . the level of exposure, whether a person was close to or far from the source of fiber release, how frequently the exposure took place and how long the exposure lasted, whether engineering or other methods of dust control were in place, and whether respiratory protection was used. The total cumulative amount of exposure should also be considered when evaluating the significance of a given subset of exposure.

(*Id.* at 73–74.) After reviewing Walls’ testimony and DePasquale’s report, Maddox concluded that Walls’ exposures to asbestos from Defendant’s products were “high, prolonged, and repetitive, and they [were] thousands of times higher than background levels.” (*Id.* at 75.) These exposures could each have caused mesothelioma, as each was greater than the “brief” exposures of “one or two days” which “have been reported to cause mesothelioma in susceptible people.” (*Id.*) Finally, like Holstein, Maddox testifies that each exposure was “a substantial contributing factor to the development of [Walls’] mesothelioma.” (Maddox rep. at 5.)

Lastly, Finkelstein reviewed studies relating to the work Walls performed during his career. He calculated that “[o]ne year as a heavy-duty truck mechanic would thus have produced an exposure of . . . 66 times his cumulative lifetime exposure from ambient air.” (Finkelstein rep. at 19.) He testifies that “Walls’ exposures to asbestos fibers from truck friction products and gaskets were substantial contributing factors in the causation of his malignant pleural mesothelioma.” (*Id.* at 27.) He lists four factors he considered “[i]n determining whether exposure to a particular defendant’s asbestos was a ‘substantial factor’

in causing” mesothelioma, including the manner of exposure, proximity to asbestos, frequency and length of exposure, and any mitigating factors. (*Id.* at 26.)

Defendants argue that these opinions are inadmissible for six reasons. First, the experts employ an “each and every exposure theory” that has been excluded by multiple federal courts. (ECF No. 477 at 7–9, 15–16, 35.) Second, the experts did not consider a lung sample analysis conducted by another of Plaintiff’s experts that revealed amphibole asbestos fibers in Walls’ lungs. (*Id.* at 5–7; June 16 Tr. at 5:3–8:9.) Third, they did not consider Walls’ time in the Navy. (ECF No. 477 at 2.) Fourth, they did not adequately account for differences in dose, fiber type, fiber length, and changes during manufacturing between “manufactured chrysotile” and other types of asbestos in finding that Walls’ exposures were significant. (*Id.* at 7, 18–20.) Fifth, they have not established at what dose chrysotile asbestos causes mesothelioma or that Walls’ exposures from any Defendant’s products exceeded that dose. (*Id.* at 20–23.) Sixth, whether an exposure is “substantial” is a question of law, not science, and they have no scientific basis for their opinion that Walls’ exposures were “substantial.” (*Id.* at 15–18.)

Based on the preponderance of evidence, the Court finds that Holstein and Maddox’s opinions that each Defendant’s products contributed to and scientifically caused Walls’ mesothelioma is relevant and based in reliable science. First, Plaintiff’s experts do not offer the “each and every exposure” theory in this case that has been rejected by other courts. The “each and every exposure” theory “represents the viewpoint that, because science has failed to establish that any specific dosage of asbestos causes injury, every exposure to asbestos should be considered a cause of injury.” *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 846 (E.D.N.C. 2015). Such testimony is distinguishable from testimony

that mesothelioma is caused by “special exposures . . . for which there is scientific evidence that the exposure increases the risk of developing” mesothelioma. *Id.* at 849. Here, Holstein and Maddox explicitly testify that Defendants’ products caused Walls’ mesothelioma because each exposure (1) contributed to his total dose and (2) *was sufficient on its own to cause mesothelioma*. Their opinions are based on reliable application of a dose-response methodology to Walls’ depositions and DePasquale’s report which, taken together, support that Walls was exposed to asbestos concentrations significantly higher than background levels from each Defendant’s products.

Second, two lung samples were taken of Walls’ lungs after he died. Defendants’ expert conducted an analysis which was shared with Holstein. (Holstein rep. III at 1–2.) This analysis found five asbestos bodies and one fiber in digested and concentrated lymph node tissue. (*Id.*) Holstein considered the report but concluded that there was not “sufficient normative data on how many asbestos fibers or bodies one would expect to find in . . . digested and concentrated lymph node tissue.” (*Id.* at 2 (emphasis omitted).) The second sample was studied by Plaintiff’s expert, Dr. Ronald Gordon. (*See* ECF No. 477–8.) Gordon found evidence of anthophyllite, amosite, crocidolite, and tremolite fibers in Walls’ lungs. (*Id.* at 4.) Plaintiff decided to withdraw Gordon as an expert and did not share his report with her experts or Defendants; however, this Court ordered her to disclose facts about the portion of Decedent’s pathology materials that Gordon analyzed (without requiring disclosure of Gordon’s opinions) on September 27, 2021, after Holstein had issued his report and two supplemental reports. (ECF No. 468 at 42–43.) Thus, Holstein did not have the benefit of Gordon’s data when issuing his reports.

Maddox did not consider either tissue sample. (Maddox rep. at 24 n.38.) However, he testified that “Lung tissue digestion . . . is a very useless means to evaluate past exposure to chrysotile . . . because the half-life for chrysotile, in the lung, is approximately one year, meaning that after 30 years, only 1/billionth of the chrysotile that was inhaled during the occupational exposure remains in the lung.” (*Id.*) He further opined that:

The primary deficiency and intellectual error in reliance upon lung fiber analysis to determine the causation of mesothelioma is that the asbestos fibers in the lung are not the fibers that caused the mesothelioma. The mesothelioma is caused by the asbestos fibers that reach the pleura—not the fibers that remain in the lung.

(*Id.*)

The fact that Holstein and Maddox did not have the benefit of Gordon’s data does weigh against the reliability of their analyses. It does not appear, however, that consideration of Gordon’s data would change either expert’s methodology or opinions. The presence of amphibole fibers in Walls’ lungs does not affect their methodology or conclusion that Walls experienced significant exposures to asbestos from Defendants’ products that contributed to his total dose and were independently capable of causing mesothelioma. Further, as Holstein testified with respect to the first lung sample, the presence of amphibole asbestos in Walls’ lungs does not refute that “Mr. Walls’ decades-long exposures to chrysotile asbestos” substantially caused his mesothelioma, since chrysotile fibers gradually disappear from view in the lungs while “amphibole fibers . . . are considerably more persistent in lung tissue.” (Holstein rep. III at 4; *see also* Maddox rep. at 25 n.38.) Thus, Holstein’s and Maddox’s failures to consider Gordon’s sample data, though concerning, do not render their opinions inadmissible.

Third, Holstein and Maddox did expressly consider Walls' experience in the U.S. Navy. (Maddox rep. at 24, ECF No. 479-7 at 273:2-15, 274:18-24 (Holstein dep.)). Although Holstein did not rule out that Walls had some exposure to asbestos while in the Navy, (Holstein dep. at 283:6-14), his review of Walls' depositions allowed him to conclude that Walls did not experience "meaningful exposures to asbestos" while in the Navy, (*id.* at 274:18-24). As discussed in more detail in Section III.B, *infra*, this analysis is consistent with record evidence that Walls had no exposure to asbestos above ambient levels while in the Navy. Maddox likewise considered Walls' naval service but reasoned that "[a]s a seaman he was always working above deck and did not have opportunity to work in the boiler or engine rooms," he "was not part of any maintenance or equipment jobs below deck (or anywhere on the ship)," and "he did not share sleeping or eating spaces with maintenance persons who worked in engine rooms." (Maddox rep. at 23.) Holstein's and Maddox's consideration of Walls' naval experience bolsters the reliability of their methodology.

Fourth, Plaintiff's experts explicitly considered dose, fiber type, and fiber length. Maddox focuses his entire specific causation testimony on chrysotile and describes in detail how chrysotile causes mesothelioma. Holstein also discusses Walls' chrysotile exposure at length. Further, as discussed above, Holstein expressly considered Defendants' evidence that chrysotile in vehicle friction products is less potent than chrysotile that has not experienced the manufacturing process. He confronted Defendants' evidence and relied on competing evidence that mechanics have contracted mesothelioma from exposure to vehicle friction products and are at an increased risk of developing mesothelioma. And Holstein considered the type of asbestos used by Defendants and that it came from vehicle friction products in his assessment of Walls' exposures to each Defendant's products.

(Holstein rep. III at 5–18.) Holstein’s consideration of these factors bolsters the reliability of his methodology.

Maddox did not grapple with possible differences between chrysotile in friction products and other forms of chrysotile. However, Plaintiff has offered evidence that asbestos in vehicle friction products is identical to the chrysotile fibers that Maddox did consider. (*See* ECF No. 479 at 9 n.23 and accompanying text.) Moreover, Defendants concede that vehicle friction products contain intact chrysotile fibers, and that no mineralogical evidence demonstrates that all chrysotile fibers lose their potency during the manufacturing process. (June 15 Tr. at 73:13–75:4.) Thus, while Maddox’s failure to consider possible differences between the potency of chrysotile in friction products and other chrysotile detracts from the reliability of his methodology, it does not require exclusion of his specific causation testimony.

Fifth, the Fourth Circuit has squarely held that precise quantification of a plaintiff’s dose “is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial exposure and need not invariably provide the basis for an expert’s opinion on causation.” *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 264 (4th Cir. 1999). Here, Defendants demand that Plaintiff establish a toxicity threshold for chrysotile that her experts say does not exist. Holstein and Maddox repeatedly testify that no study reliably establishes a quantifiable toxicity threshold and that, in their opinion, every significant exposure to asbestos contributes to cause cancer. Further, Holstein and Maddox testify that different individuals have varied susceptibility, meaning any defined threshold could vary from person to person. To establish causation, therefore, they do not attempt to establish threshold toxicity, but rather qualitatively assess Walls’ exposure to asbestos from

Defendants' products using his testimony and DePasquale's report and compare those exposures to other exposure levels that have been found to cause mesothelioma. The Court finds that this qualitative assessment is consistent with Fourth Circuit precedent and sufficiently reliable in this case to be admitted.

Accordingly, the Court finds that the portion of Holstein and Maddox's specific causation testimony concluding that each Defendant's products contributed to Holstein's mesothelioma is admissible.

Holstein and Maddox's "substantial factor" testimony, on the other hand, is inadmissible. "Substantial factor" is a legal standard, not a scientific one. According to Holstein, even brief or low-level exposures to asbestos may create, promote, or shorten the latency period of cancer, (Holstein rep. I at 27), but not "every one of the great number of events" that contributes to mesothelioma constitutes a "substantial factor." *See Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162 (1986) (quoting Restatement (Second) of Torts § 431 cmt. a. (Am. Law Inst. 1965)). Rather, this legal standard combines scientific causation with "the idea of responsibility." *Id.* The substantial factor test requires that a defendant who causes a plaintiff to have no more than a "casual or minimum" exposure is not held joint and severally liable for a resulting injury, particularly when that defendant's responsibility for the injury is "dwarfed" by another potential cause. *See Connor*, 996 F.3d at 149, 155. Here, Holstein and Maddox offer no scientific standard in this case to support their conclusions that some exposures constituted a "substantial factor" beyond a conclusory listing of the relevant *Lohrmann* factors. Moreover, their testimony could confuse the jury by suggesting that "substantial factor" is a scientific threshold rather than a legal determination of responsibility. The Court therefore finds that Holstein and Maddox

may testify that Defendants' products scientifically contributed to and caused Plaintiff's mesothelioma, but they will be precluded from testifying that any exposure was a "substantial factor," as such testimony will not aid the jury and could usurp the jury's role by implying that "substantial factor" is a scientific standard, rather than a legal standard.

Finally, Plaintiff has failed to show based on the preponderance of evidence that Finkelstein may testify to specific causation in this case. Unlike Holstein and Maddox, Finkelstein spends very little time in his report explaining his method for determining whether an exposure contributes to or causes mesothelioma. He lists several relevant factors and relevant facts from Walls' testimony but does not clearly apply the factors to the facts or otherwise explain his causation testimony. He does not discuss the impact of Walls' tissue samples or naval service. Although he estimates Walls' total annual exposure level, he does not evaluate exposure to Defendants' products individually. Accordingly, Finkelstein will be permitted to testify to general causation and that Walls' cumulative dose as a truck mechanic was sixty-six times higher each year than his total lifetime exposure to ambient background air, but he will not be permitted to testify that Defendants' products contributed to, caused, or were a substantial factor in causing Walls' mesothelioma.

In conclusion, Defendants' motions will be granted in part and denied in part. Each expert will be permitted to offer general causation testimony. Holstein and Maddox will be permitted to opine that each Defendant's products scientifically contributed to and caused Walls' mesothelioma. Finkelstein will not be permitted to testify that Defendants' products caused Walls' mesothelioma. No expert will be permitted to testify that a product constituted a "substantial factor" in causing Walls' mesothelioma.



#### **IV. PLAINTIFF'S MOTIONS (ECF Nos. 264; 262; 281; 255; 253)**

##### **A. Naval Exposures (ECF No. 264)**

Turning to Plaintiff's motions, Plaintiff first seeks to exclude "evidence of occupational or bystander exposure to asbestos in the US Navy" and "Defense Naval Researchers." (ECF No. 264.)

Defendants forecast that they will argue to the jury that Walls' mesothelioma was not caused by his exposure to asbestos from their products by offering evidence that Walls was exposed to asbestos while in the Navy. Defendants offer Captain Margaret McCloskey and Christopher Herfel as experts on naval engineering. (ECF Nos. 350-3 ("Herfel rep."); 350-5 ("McCloskey rep.")) McCloskey is a Captain in the U.S. Navy and holds a master's degree in mechanical engineering. (McCloskey rep. at 1-2.) Her two decades of service include supervising repairs and upgrades of numerous naval vessels while serving as an Asbestos Control Officer, Chief Engineer, and Assistant Chief of Staff in the Aircraft Carrier Ship Maintenance and Material Readiness Office. (*Id.* at 2-5.) She currently serves as an archival researcher for naval ships, ship systems, and ship operations. (*Id.* at 5.) Herfel holds a Bachelor of Science in Marine Engineering and a Master of Business Administration. (Herfel rep. at 1.) He served as an Officer in the U.S. Naval Reserve for eight years and managed repair and overhaul of military and commercial ships as a Ship Superintendent in a Maryland shipyard. (*Id.*) He has spent the past seventeen years researching "U.S. Navy policy, practices, doctrine and procedures regarding the materials used to construct, maintain and repair U.S. Navy ships." (*Id.*)

McCloskey reviewed documents from the National Archives and Records Administration to form opinions about the types, composition, amounts, and location of

asbestos containing materials on naval vessels, as well as the “job responsibilities, training, and duties of enlisted Sailors.” (McCloskey rep. at 5–6.) Herfel reviewed Navy records relating to the design, construction, operational employment, and overhaul of the vessels on which Walls served; records relating to the duties, responsibilities and training of seamen and torpedoman; and Walls’ deposition testimony. (Herfel rep. at 2.) Their proffered opinions include the following: (1) Walls’ naval work “put him in close proximity to others working with asbestos-containing” materials, which “presented a great potential for exposure”; (2) Walls’ daily cleaning details “likely would have included cleaning and disposing of insulation debris removed during routine daily maintenance and shipyard overhaul and repair periods”; (3) “[s]everal tons of amosite asbestos-containing materials were used in the construction, conversion, maintenance, modification, overhaul, and repair of” ships on which Walls served; (4) Walls “was likely on board” when his ship underwent an overhaul that would have produced “[l]arge amounts of thermal and anti-speat insulation dust”; (5) “[a]sbestos dust could also be created by normal shipboard operations, shock and vibration from firing of the ship’s weapons and external environmental forces, all of which likely occurred” during Walls’ service; (6) the Navy identified torpedomen like Walls “as the 17th most likely group of Sailors to develop asbestos-related diseases”; and (7) the U.S. Department of Veterans Affairs currently considers sailors who served on ships built prior to 1983 “to be at risk for asbestos exposure.” (McCloskey rep. at 6; Herfel rep. at 26–27.)

Plaintiff argues that these opinions are irrelevant or not based on sufficient facts or data because no evidence supports that Walls was actually exposed to any asbestos while in the Navy above background levels. (ECF No. 267 at 11–18; June 15 Tr. at 6:16–21:13.) Because no expert obtained Walls’ service record, (*see* Herfel rep. at 3), the only direct

evidence of Walls' activities while serving in the Navy come from his unrebutted deposition testimony. As McCloskey conceded in deposition, nothing in Walls' testimony supports that he worked with any asbestos containing products while in the Navy. (ECF No. 267-13 at 45:22–25, 74:5-10.) According to Walls, he served as a seaman and torpedoman aboard two ships during his short naval career. Each ship was longer than a football field at 391 and 491 feet, and each had numerous decks. (June 15 Tr. at 8:10-21, Pl. Ex. 1 at 9.) Walls' living spaces were insulated with cork, not asbestos, (ECF No. 521-10 § L-2-a), and he never worked with, ate with, slept near, or otherwise meaningfully interacted with sailors who worked with asbestos, (ECF Nos. 268-1 at 26:1-4, 28:11-22; 268-2 at 100:9-13; 268-3 at 28:5-19). Although he did help clean above deck, his unrebutted testimony shows that he never cleaned up insulation, never spent time with sailors who worked with insulation, and never observed anyone disturb insulation of any kind. (*See* ECF No. 268-3 at 29:3-18.)

The Court agrees that McCloskey's and Herfel's opinions that Walls was exposed to asbestos during the Navy are speculative and not based on sufficient facts or data. McCloskey's opinion that Walls' naval work "put him in close proximity to others working with asbestos-containing" materials is directly contradicted by Walls' testimony. Herfel's opinions that Walls "*likely*" cleaned insulation, "was *likely* on board" during a ship overhaul and was "*likely*" exposed to asbestos during the firing of weapons are all conjectural and not based on any evidence of actual exposure. Accordingly, testimony detailing the locations of asbestos on each vessel is irrelevant as there is no evidence that Walls ever interacted with asbestos in these locations.

McCloskey's and Herfel's remaining testimony merely establishes that asbestos was present on Walls' ships. Neither expert is qualified as an industrial hygienist to offer any

opinion as to the level of Walls' exposure from background levels, and Defendants explicitly state that "McClosky [and] Herfel . . . will not opine as to the cause of [Walls'] mesothelioma." (ECF No. 350 at 5.) Plaintiff, however, concedes that asbestos was present aboard Walls' ships, and that Walls was exposed to some background level of asbestos while in the Navy. (*See* June 15 Tr. at 16:11-13.) Thus, McCloskey's and Herfel's remaining testimony is duplicative and will not aid the jury in determining a fact in issue.

Finally, Herfel's testimony relaying that the Navy and Department of Veterans Affairs consider torpedomen and sailors who served on ships built prior to 1983 to be at risk for asbestos exposure and asbestos-related disease is offered without context, and he is not qualified to opine on the level of risk associated with Walls' service in the Navy.

To the extent Plaintiff also seeks to exclude all other, non-expert evidence concerning Walls' experience in the Navy, such motion will be denied. Defendants are correct that they may offer relevant evidence of potential exposure to asbestos Walls sustained while serving in the Navy that is not substantially more prejudicial than probative, *see* Fed. R. Evid. 403, and Plaintiff bears the burden to show that exposure to Defendants' products was the substantial cause of Walls' mesothelioma, *Finch*, 972 F.3d at 512.

Accordingly, Plaintiff's motion will be granted in part and denied in part. McCloskey's and Herfel's expert testimony will be excluded. Plaintiff's motion will be denied with regard to non-expert evidence of asbestos exposure to Walls in the Navy.

**B. Cross-Examination about “Manufactured Chrysotile” (ECF No. 262)**

Plaintiff next requests “that this Court prevent Defense counsel from cross-examining Plaintiff’s Experts about whether the manufacturing process alters chrysotile within friction products.”<sup>5</sup> (ECF No. 431 at 4–5.)

This motion is not a true *Daubert* motion. Cross-examination questioning and evidence is not governed by *Daubert* and Rule 702, which only govern the admissibility of expert opinion. *See* Fed. R. Evid. 702. Relevant evidence on cross-examination may be admitted so long as it is not substantially more prejudicial than probative. Fed. R. Evid. 402, 403. Here, Plaintiff’s motion does not seek to exclude Defendants’ experts’ testimony, but rather seeks to preclude Defendants’ attorneys from questioning her experts’ about “manufactured chrysotile.” (ECF Nos. 262; 431 at 4–5.)

Plaintiff has offered evidence challenging the probative value of questions about “manufactured chrysotile.” She offers evidence that unaltered chrysotile fibers are found in vehicle friction products, (ECF No. 431 at 2 n.3–5), and her experts testify that exposure to vehicle friction products causes mesothelioma, *see* Section III.A.1, *supra*. However, she has not shown that cross-examining her experts on these topics would be substantially prejudicial. It appears that Plaintiff’s experts can mitigate any prejudice by explaining their

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<sup>5</sup> Plaintiff initially moved to exclude “any testimony that the chrysotile in brakes was converted to forsterite in the manufacturing process.” (ECF No. 262 at 1.) Forsterite is a harmless substance created by heating chrysotile to extremely high temperatures. (*See* ECF Nos. 263 at 2, 6; 263-3 at 229.) Defendants, however, responded that “manufactured chrysotile fibers may be rendered inert during the brake manufacturing process, long before they are converted to forsterite.” (*See, e.g.*, ECF No. 379 at 17.) Plaintiff then clarified in her Reply that her motion seeks to exclude any evidence on cross-examination that the manufacturing process alters chrysotile within friction products. (ECF No. 431 at 4–5.) “Manufactured chrysotile” was the focus of the Parties’ arguments during the June 15, 2022, hearing on this motion, and Defendants did not object to inclusion of this evidence in Plaintiff’s motion. (*See* June 15 Tr. at 37:19-25, 56:1-3, 58:12–62:14.)

opinions on “manufactured chrysotile.” As discussed above, whether Plaintiff’s experts considered possible differences in potency between chrysotile in friction products and other chrysotile is relevant to their reliability and credibility. Thus, Plaintiff’s motion will be denied.

**C. Testimony that Exposure to Vehicle Friction Materials Cannot Cause Mesothelioma (ECF No. 281)**

Plaintiff next moves to exclude “any testimony that a vehicle mechanic[’s] exposures to chrysotile friction products cannot cause mesothelioma.” (ECF Nos. 281 at 1; 282 at 1.)

As discussed in Section III.A.1, *supra*, the parties’ experts disagree on general causation—whether chrysotile fibers in vehicle friction products can cause mesothelioma. The Parties do not identify in their briefing, however, which of Defendants’ experts offer the challenged general causation opinion. (*See generally* ECF Nos. 282; 379 at 26–33; 429.) At oral argument, Plaintiff argued that her motion affects nine Defense experts. (June 15 Tr. Pl. Ex. 4 at 4.) However, while it appears that some of Defendants’ experts seek to testify that exposure to vehicle friction products categorically cannot cause mesothelioma,<sup>6</sup>

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<sup>6</sup> *See, e.g.*, ECF Nos. 292-9 at 65 (Mowat rep.) (“[A]ny exposure to asbestos that Mr. Walls may have experienced when performing vehicle maintenance and repair work during his career . . . would not have put him at increased risk for developing mesothelioma.”); 344-3 at 2 (Oury rep. I) (“[A]ny exposure to automotive friction products would not have contributed to the development of Mr. Walls’ mesothelioma as individuals occupationally exposed to friction products fail to show evidence of asbestos accumulation in their lungs and do not have elevated risks for asbestos associated diseases.”); 345-1 at 25 (Crapo rep.) (opining that “brakes and/or engine gaskets [which] contained chrysotile asbestos in a resin matrix . . . would result in only low level chrysotile exposures that would not create a risk for disease” and “[f]ull time automotive mechanics and brake workers have been found to not be at an increased risk for development of mesothelioma”); 345-7 at 18 (Alexander rep.) (“Since the epidemiologic evidence does not support a conclusion that motor vehicle repair work places people at increased risk of mesothelioma, there is no epidemiologic bases for a conclusion that Mr. Walls’ motor vehicle repair work . . . increased his risk of developing mesothelioma.”).

others testify that such products do not increase the risk of developing mesothelioma *at low levels*,<sup>7</sup> and some appear to testify only in the negative that no studies have shown that exposure to vehicle products causes mesothelioma.<sup>8</sup> Identifying which experts intend to offer the challenged opinion is critical to conducting a *Daubert* analysis because the Court can only rule on the scientific validity of *methodologies*, not particular conclusions. *Daubert*, 509 U.S. at 595 (“[A court must focus] solely on principles and methodology, not on the conclusions that they generate.”). Experts with different qualifications and who employ different methodologies must be evaluated separately. Thus, this Court will evaluate Plaintiff’s motion with respect to the testimony of Dr. Tim Oury, Dr. Dominik Alexander, Dr. Fionna Mowat, and Dr. James Crapo, the experts who appear to offer the challenged opinion. The Court will deny without prejudice Plaintiff’s motion as it relates to Defendants’ remaining experts, and Plaintiff may raise her objection at trial if another expert is offered to testify that mechanics categorically cannot contract mesothelioma from vehicle friction products.

Oury, a pathologist,<sup>9</sup> authored his initial report on July 8, 2020, (ECF No. 344-3 (Oury rep. I)), and submitted a supplemental report on April 14, 2021, (ECF No. 345-5

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<sup>7</sup> See, e.g., 259-2 at 26 (Sahmel rep.) (“Walls’ cumulative chrysotile asbestos exposure potential associated with brake or clutch work [supplied by certain Defendants was] well within the range of cumulative lifetime background exposures to the general U.S. population, [which] have not been shown to increase the risk of asbestos related disease, including mesothelioma.”).

<sup>8</sup> See, e.g., ECF No. 393-13 at 111 (Finn rep.) (“No scientific literature currently exists linking chrysotile-related brake exposures with asbestos-related mesothelioma.”).

<sup>9</sup> Oury holds an M.D. and Ph.D. in Pathology-Biochemistry from Duke University. (ECF No. 344-1 at 2.) He is a Professor of Pathology at the University of Pittsburgh and Co-Director of the university’s Pathologist Investigator Residency-Research Training Program. (*Id.*) He has authored over one hundred publications, including on asbestos related disease. (*Id.* at 4–18.) The Court finds that Oury is qualified as an expert in pathology.

(Oury rep. II)). His initial one-page report summarizes his study of Walls' lung tissue sample. (Oury rep. I at 1.) He states that "insufficient lung tissue was available to determine . . . if asbestos did or did not contribute to the pathogenesis of Mr. Walls' mesothelioma." (*Id.*) He then reports without citation or explanation that "any exposure to automotive friction products would not have contributed to the development of Mr. Walls' mesothelioma as individuals occupationally exposed to friction products fail to show evidence of asbestos accumulation in their lungs and do not have elevated risks for asbestos associated diseases." (*Id.*) In his supplemental report—also one page—Oury details his and another laboratory's closer inspection of Walls' tissue. (Oury rep. II at 1.) He states that these inspections "indicate[ ] that prior exposure to amphibole asbestos is the cause of Mr. Walls' mesothelioma." (*Id.*)

The Court finds that Oury has failed to show that his conclusion that "individuals occupationally exposed to friction products . . . do not have elevated risks for asbestos associated diseases" is based on his expertise as a pathologist or any reliable scientific methodology. The conclusory opinion is stated without explanation or citation in his initial report and is not repeated in his supplemental report. The supplemental report draws a conclusion about Walls from his lung tissue rather than from a categorical assumption about vehicle friction products and mechanics' exposures. Thus, Plaintiff's motion will be granted as to Oury.



The remaining experts submit detailed reports that root their conclusions primarily in epidemiology. Alexander, an epidemiologist,<sup>10</sup> testifies that “epidemiologic evidence is not supportive of an independent association between exposure to chrysotile asbestos fibers and risk of mesothelioma,” and that “[t]he weight of the epidemiologic evidence does not support an increased risk of mesothelioma among motor vehicle mechanics.” (Alexander rep. at 15–16.) He bases these conclusions primarily on his own meta-analysis of sixteen epidemiologic studies, which he discusses in his report. (*Id.* at 16–17.) He also identifies and distinguishes epidemiologic studies that contradict his conclusion. (*Id.* at 15.)

Mowat, a biomedical engineer and expert in risk assessment,<sup>11</sup> bases her conclusion that “any exposure to airborne asbestos that Mr. Walls may have experienced while performing vehicle repair and maintenance work . . . did not contribute to his risk for developing mesothelioma” on two areas of scientific research. (Mowat rep. at 56.) First, like Alexander, she reports that “[e]pidemiologic studies of automobile mechanics, vehicle repair workers, and garage mechanics/workers . . . have shown repeatedly that these workers are not at an increased risk for developing mesothelioma.” (Mowat rep. at 24.) She lists the studies upon which she relies, discusses in detail meta-analyses of those studies, and discusses criticisms of studies that come to opposite conclusions. (*Id.* at 24–28.)

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<sup>10</sup> Alexander is the President and Principal Epidemiologist with an epidemiology and health research consulting firm. (ECF No. 355-3 at 2 (Alexander rep.)). He holds a Ph.D. in Epidemiology from the University of Alabama-Birmingham School of Public Health and a Master of Science in Public Health in Epidemiology and Biostatistics from the University of South Florida College of Public Health. (*Id.* at 37.) He has authored numerous published, peer-reviewed articles. (*Id.* at 40–45.) The Court finds that Alexander is qualified as an expert in epidemiology.

<sup>11</sup> Mowat is a Principal Scientist, Corporate Vice President, and Director of the Health Sciences Center with a scientific research and consulting firm. (Mowat rep. at 3.) She holds a Ph.D. in Biomedical Engineering from Tulane University. (*Id.*) She has studied risk assessment and asbestos for two decades and has published or co-authored numerous asbestos exposure simulations. (*Id.*) The Court finds that Mowat is an expert in biomedical engineering and risk assessment.

Second, she details historic measurements of exposures to mechanics and concludes that these exposures “were well below the current regulatory PEL for asbestos, both on an eight-hour TWA and cumulative exposure basis.” (*Id.* at 56.)

Finally, Crapo, a pulmonologist,<sup>12</sup> likewise relies on meta-analysis of epidemiological studies to testify that “[t]he conclusions from this meta-analysis . . . were that motor vehicle mechanics . . . are not at an increased risk of mesothelioma.” (*Id.* at 11.) In addition, he bases his conclusions on a series of studies that exposed rats to chrysotile asbestos. (*Id.*) The study found that rats, when exposed to the equivalent of background ambient levels experienced by people in major U.S. cities over their lifetime had no lung inflammation after one year. (*Id.*) Finally, he describes the findings of various studies and concludes that “[i]n populations exposed predominately to chrysotile, mesothelioma either does not occur or the relatively few cases that do occur have exposure histories in the hundreds of fiber years or can be shown by careful study to have likely had a concurrent amphibole exposure.” (*Id.* at 13.) Taking this together, he testifies that “it is possible that (pure) chrysotile is nonpotent for causation of mesothelioma,” and if it is potent, “chrysotile can be a cause for pleural mesothelioma at high doses,” but that Walls’ exposures did not reach those high levels. (*Id.* at 14–15, 25.)

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<sup>12</sup> Crapo is a licensed pulmonologist and Professor of Medicine at National Jewish Health and at the University of Colorado. (Crapo rep. at 1–2.) He holds a medical degree from the University of Rochester Medical School and previously worked at the Environmental Toxicology Branch of the National Institute of Environmental Health Sciences, where he “conducted extensive research on the subject of lung disease and the effects of asbestos on the lungs.” (*Id.* at 1.) He has served as Chief of Pulmonary Medicine at Duke University and Associate Dean at the University of Colorado. (*Id.*) He served on the EPA’s Clean Air Scientific Advisory Committee. (*Id.* at 2.) The Court finds that Crapo is qualified as an expert in pulmonology.

Plaintiff argues that the epidemiological studies, meta-analyses, and animal studies are methodologically flawed and do not fit the facts of this case. (ECF No. 282 at 12–27.) First, she argues that each expert failed to address a 2018 study of textile workers in China that found low levels of exposure to chrysotile asbestos increases the risk of developing mesothelioma. (*Id.* at 15.) Second, she argues that not all vehicle mechanics have similar experiences because brake, clutch, and gasket work is not consistently performed, and epidemiological studies relied on by Defendants’ experts do not account for these variations. (*Id.* at 20–21.) Third, she argues that the rat study only studied lung inflammation and not cancer, and one year is an insufficient amount of time to determine whether rats could develop cancer after being exposed to chrysotile asbestos. (*Id.* at 25–26.)

The Court finds that Alexander and Mowat have a sufficiently reliable epidemiological basis to testify that exposure to vehicle friction products does not cause mesothelioma. These experts are qualified to assess the validity of epidemiological studies and meta-analyses and describe in sufficient detail the findings of relevant studies that support their conclusions. Plaintiff’s criticisms of the underlying studies are not without merit, and it is of concern that the studies may include mechanics who had lower exposures to asbestos than Walls. It appears, however, that Alexander’s and Mowat’s conclusions are based on some of the best available studies, and Plaintiff’s contentions can be presented to the jury through cross-examination. Thus, the Court finds that Defendants have met their burden to show that, based on the preponderance of the evidence, Alexander’s and Mowat’s conclusions are based on a reliable assessment of available epidemiological studies of vehicle mechanics.

Crapo's testimony requires a slightly different result. Crapo, a pulmonologist, is less qualified to opine on the meaning and reliability of epidemiological studies than his colleagues. He explains that "it is *possible* that (pure) chrysotile is nonpotent," but does not state, as Mowat and Alexander do, that chrysotile from vehicles is, in fact, nonpotent. (Crapo rep. at 14 (emphasis added).) The cited animal study does not support his categorical conclusion either, as the study measured background levels of chrysotile asbestos and only documented changes in inflammation after one year, not long-term development of cancer. Crapo therefore will be precluded from testifying that vehicle friction products categorically cannot cause mesothelioma. Plaintiff has not challenged Crapo's testimony that Walls' exposure was too low to cause mesothelioma, and the Court expresses no opinion on the admissibility of such testimony.

Accordingly, Plaintiff's motion will be granted as to Oury and Crapo but denied as to Alexander and Mowat.

**D. No Observed Adverse Effect Level (ECF No. 255)**

Plaintiff next moves to preclude Defendants' experts from testifying that cumulative exposures to chrysotile asbestos that do not exceed 208 to 415 f/cc-years do not increase a person's risk of mesothelioma. (ECF Nos. 254 at 1; 255.)

Several of Defendants' experts seek to testify about a 2008 study, updated in 2016, that "evaluated the no-observed adverse effect level for chrysotile asbestos and for mesothelioma" and "determined it to be between 208 and 415 fiber·cc·years." (*See, e.g.*, Crapo rep. at 24.) In other words, the study found that a person could be exposed to a cumulative annual exposure of up to between 208 and 415 fibers per cubic centimeter before experiencing an observable adverse reaction. (*See* June 15 Tr. at 106:22–107:3.)

Plaintiff argues that the underlying study is methodologically flawed. First, the 2008 meta-analysis is based on only four studies, and the 2016 update is based on only three. (*Id.* at 107:10-11, 113:5-6.) Second, both the meta-analysis and update purportedly excluded any study where amphibole asbestos represented 10% or more of the cumulative exposure of the individuals in the study, but actually included one study wherein 11.4% of individuals' exposure was to amphiboles. (*Id.* at 107:12-17.) Third, Plaintiff argues that the underlying studies did not actually measure asbestos fibers/cc and instead inaccurately estimated asbestos exposure based on the total particles of dust per cubic foot, asbestos or otherwise. (*Id.* at 109:1–112:4.) Fourth, the meta-analysis is based on outdated data, as several cases of mesothelioma developed in the studied cohorts after the meta-analysis was originally published. (*Id.* at 114:13–115:15.) Fifth, the 2016 updated meta-analysis inexplicably excluded the study with the highest cancer rate from the meta-analysis, inflating the estimated no observed adverse effect level. (*Id.* at 113:9-13.) Finally, Plaintiff cites a 2018 study not included in the meta-analysis of textile workers who were exposed almost exclusively to chrysotile asbestos and found a significantly increased risk of mesothelioma at just 0–0.1 fiber/cc/years. (*Id.* at 114:2-12.)

Defendants do not directly respond to these criticisms. Instead, they argue that the subject study is just one way that their experts “check[] their work to say, yes, these exposures . . . are not sufficient to cause an increased risk of mesothelioma.” (*Id.* at 116:17-20.) Defendants' experts do not use the challenged study “for mathematical precision,” but rather as support to say that the dose-response threshold is “high, several orders of magnitude higher than mechanic exposure.” (*Id.* at 118:6–119:13.) Defendants argue that

“the precise number, whether it is 200 or 100 . . . it doesn’t really matter because we are so far apart from Mr. Walls’ mechanic exposures.” (*Id.* at 119:24–120:3.)

The Court finds that mathematical precision does matter. “The main purpose of *Daubert* exclusion is to protect juries from being swayed by dubious scientific testimony.” *Nease v. Ford Motor Co.*, 848 F.3d 219, 231 (4th Cir. 2017). Numbers can be particularly persuasive as they imply a certain level of exactness and reliability. *See E. Auto Distribs., Inc. v. Peugeot Motors of Am., Inc.*, 795 F.2d 329, 338 (4th Cir.1986) (“Scrutiny of expert testimony is especially proper where it consists of an array of figures conveying a delusive impression of exactness in an area where a jury’s common sense is less available than usual to protect it.”). But here, Defendants seek to introduce quantitative testimony to make a qualitative point—to dramatize to the jury the large difference between Walls’ exposures and the high exposure levels found to cause no adverse effect. Rule 702 and *Daubert* directly prohibit this testimony.

Defendants have failed to meet their burden to show that the measures in question are based on a scientifically reliable methodology. Consequently, their experts will be precluded from offering these numbers to the jury.

#### **E. Quantification of Relative Asbestos Fiber Potency (ECF No. 253)**

Finally, Plaintiff moves to preclude Defendants’ experts from quantifying the difference in potency between chrysotile and other asbestos fibers. (ECF No. 253.)

As mentioned in Section III.A.1, *supra*, the parties agree that chrysotile asbestos is less potent than amphibole asbestos but disagree on how to quantify that potency ratio. (*See* June 15 Tr. at 130:8-11.) Defendants’ experts cite meta-analysis studies that estimate amphiboles to be as much as 1000 times more potent than chrysotile. (*See, e.g.*, Crapo rep.

at 14.) Plaintiff cites other studies that estimate amphiboles generally to be only three times more potent than chrysotile, and others that estimate the potency ratio between crocidolite and chrysotile to be anywhere from 4:1 to 500:1, and amosite to chrysotile to be anywhere from 1:1 to 100:1. (ECF No. 260 at 3–4.)

Plaintiff argues that the meta-analysis studies cited by Defendants are unreliable and, therefore, Defendants’ estimated quantifications based on those studies are likewise unreliable. First, she argues that the underlying studies relied upon by the meta-analysis made flawed assumptions about the exposure level of the studied individuals and is based on outdated data. (June 15 Tr. at 131:5–132:18 (calling its calculations “guesstimates”).) Seven of eight cases of mesothelioma in one cohort developed after the corresponding study was published and is not accounted for in its results; in another, all nine cases of mesothelioma developed after the corresponding study’s publication. (*Id.* at 133:25–135:8.) Second, she argues that the authors of one relied upon study acknowledge that their findings are based on limited data and “should be considered a proof of concept, more than a final result.” (*Id.* at 135:9-25.) This study has received criticism from medical associations and the chair of the EPA asbestos subcommittee. (*Id.* at 136:12-17.)

As in Section III.E, *supra*, Defendants do not directly respond to these criticisms. They acknowledge that the challenged studies are “estimates” but contend that they are “not relied upon by defense experts for mathematical precision.” (*Id.* at 139:9-16.) They argue that mathematical precision of the estimates “doesn’t matter . . . because Mr. Walls’ exposure to chrysotile were so slight.” (*Id.* at 153:18-24.) Instead, they argue that the quantitative estimates, even if inaccurate, are useful to provide “context” to the jury about

“what is the potency difference” and whether “we are talking about a little bit of difference or a lot of difference.” (*Id.* at 139:9-16.)

As above, Defendants’ arguments are entirely unpersuasive. Rule 702 and *Daubert* exist precisely to prevent experts from offering opinions that *seem* scientific but are not based on any reliable method. Numbers can be particularly persuasive, and this Court as gatekeeper must ensure that the jury is not led astray by numbers that have no reliable scientific basis. Defendants have not met their burden to show that their experts have accurate numerical answers to “what is the potency difference” or whether the difference is “a little bit” or “a lot of difference.” Because Defendants have not met their burden, Plaintiff’s motion will be granted, and Defendants’ experts will be precluded from testifying to any numerical difference in potency between asbestos fiber types.

### **CONCLUSION**

The parties’ experts have competing views about the capacity of chrysotile asbestos used in Defendants’ products to cause mesothelioma generally and the specific cause of Walls’ mesothelioma in this case. The Court finds that these competing theories are relevant and based on reliable scientific methodologies, with exceptions detailed above. The question of whether Defendants’ products caused Walls’ injury is a question of fact for the jury, and the jury will benefit from hearing opposing scientific evaluations that are based on competing but reliable scientific methodologies.

For the reasons stated herein, the court enters the following:

### **ORDER**

**IT IS THEREFORE ORDERED** that Defendants’ Motion to Exclude the Testimony of Dr. Finkelstein, (ECF No. 307), is **GRANTED** in part and **DENIED** in



part. The motion is **GRANTED** with respect to Finkelstein's testimony concerning specific causation. Defendants' motion is **DENIED** with respect to Finkelstein's testimony concerning general causation.

**IT IS FURTHER ORDERED** that Defendants' Motion to Exclude the Testimony of Dr. Holstein, (ECF No. 308), is **GRANTED** in part and **DENIED** in part. The motion is **GRANTED** with respect to Holstein's testimony that Defendants' products were a "substantial factor" in causing Walls' mesothelioma. Defendants' motion is otherwise **DENIED**.

**IT IS FURTHER ORDERED** that Defendants' Motion to Exclude the Testimony of Dr. Maddox, (ECF No. 309), is **GRANTED** in part and **DENIED** in part. The motion is **GRANTED** with respect to Maddox's testimony that Defendants' products were a "substantial factor" in causing Walls' mesothelioma. Defendants' motion is otherwise **DENIED**.

**IT IS FURTHER ORDERED** that Plaintiff's Motion to Exclude Evidence of Occupational or Bystander Exposure to Asbestos in the US Navy and to Exclude Defense Naval Researchers, (ECF No. 264), will be **GRANTED** in part and **DENIED** in part. The motion is **GRANTED** with respect to Defendants' naval researchers, and Christopher P. Herfel and Captain Margaret McClosky will be excluded from trial. The motion is otherwise **DENIED**.

**IT IS FURTHER ORDERED** that Plaintiff's Motion to Exclude Any Testimony that the Chrysotile in Brakes Was Converted to Forsterite in the Manufacturing Process, (ECF No. 262), is **DENIED**.

**IT IS FURTHER ORDERED** that Plaintiff's Motion to Exclude Any Testimony that a Vehicle Mechanics Exposures to Chrysotile Friction Products Cannot Cause Mesothelioma, (ECF No. 281), is **DENIED**.

**IT IS FURTHER ORDERED** that Plaintiff's Motion to Exclude Reference to and Reliance upon an Alleged No Observed Adverse Effect Level for Exposure to Chrysotile Asbestos and Mesothelioma, (ECF No. 255), is **GRANTED**.

**IT IS FURTHER ORDERED** that Plaintiff's Motion to Prohibit Reference to and Reliance upon Speculative Estimates of Quantitative Relative Asbestos Fiber Potency, (ECF No. 253), is **GRANTED**.

This, the 11<sup>th</sup> day of August 2022.

/s/ Loretta C. Biggs  
United States District Judge